Refine Search

Search Results -

Terms	Documents
hydrangeae	24

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L3

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Wednesday, December 21, 2005 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L3	hydrangeae	24	L3
L2	L1 and hydrangeae	0	L2
L1	(alcohol or ethanol) same liver same peroxidation	36	L1

END OF SEARCH HISTORY

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L1: Entry 2 of 36 File: USPT Aug 30, 2005

DOCUMENT-IDENTIFIER: US 6936424 B1
TITLE: Materials and methods for detection and treatment of breast cancer

Other Reference Publication (11):
Aleynik et al., "Increased circulating products of lipid peroxidation in patients with alcoholic liver disease," Alcohol Clin. Exp. Res., 22(1):192-196 (Feb. 1998).

Previous Doc Next Doc Go to Doc#

L1: Entry 3 of 36 File: USPT Nov 9, 2004

DOCUMENT-IDENTIFIER: US 6814951 B1
TITLE: Acetaldehyde and malondialdehyde protein adducts as markers for alcohol liver disease

Brief Summary Text (6):
Another reactive aldehyde involved in alcohol liver injury is malondialdehyde (MDA). Malondialdehyde is formed by the peroxidation of polyunsaturated fatty acids and from the oxidative degradation of deoxyribose by a hydroxy radical. MDA is also produced in mammalian tissues as a side product of prostaglandin and thromboxane biosynthesis.

Brief Summary Text (7):
Several studies have suggested that chronic ethanol consumption induces hepatic lipid peroxidation which in turn, generates malondialdehyde. MDA is toxic, mutagenic, and inactivates enzymes due to modification of lysine residues. MDA protein adducts have been detected in the liver following administration of agents that promote lipid peroxidation such as carbon tetrachloride, iron overload, and more recently chronic ethanol feeding. It has been shown to form an adduct with a lysine residue (.epsilon.-amino group) of proteins and that MDA reacts with a primary amine to give a 1:1 Schiff base. For further information about MDA protein adducts see Houglum et al., J. Clin. Invest., 86: 1991 (1990) incorporated herein by reference.

Brief Summary Text (10):
It is yet another object of the invention to provide a marker for alcohol liver damage which can be used to indicate the presence of liver disease, or other diseases with increased lipid peroxidation, lipids and/or or acetaldehyde which can include but is not limited to atherosclerosis or fat content for animals.

Brief Summary Text (19):
In addition to the above-identified novel hybrid adducts, the adducts of the invention have several important immunological properties which can be exploited for further chemical and immunological assay procedures. Monoclonal and polyclonal antibodies have been produced which recognize these adducts and can be used to identify them as markers of alcohol liver disease or other diseases associated with increased lipid peroxidation, lipids, and/or acetaldehyde such as atherosclerosis and fat content for domestic animals.

Detailed Description Text (82):
Numerous studies in the literature have applied immunochemical techniques to indicate the presence of a variety of protein adducts in the livers of ethanol-treated animals. These would include acetaldehyde adducts, MDA adducts, and more recently hydroxyethyl radical-derived adducts. However, structural information and epitope characterization of these adducts are lacking, and quantitative data have not been reported. In contrast, the applicants have provided quantitative estimates for MAA adduct formation and proposed structures of the MAA adducts. Furthermore, the results indicate that MDA and acetaldehyde react together in a synergistic manner which demonstrates that MAA adduct formation would be favored over adducts formed with acetaldehyde of MDA alone and that MAA adducts may represent a major species of adducts formed in the liver during ethanol metabolism in vivo. Since

both the covalent binding of acetaldehyde to proteins and increased lipid peroxidation have been proposed as possible mediators of ethanol-induced liver injury, MAA protein-adduct formation represents an event dependent on both mechanisms, suggesting a common or unifying process (i.e. MAA adduct formation) by which both mechanisms can contribute to alcohol hepatotoxicity.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L1: Entry 6 of 36 File: USPT Dec 17, 2002

DOCUMENT-IDENTIFIER: US 6495170 B1
TITLE: Method of increasing the presence of glutathione in cells

Brief Summary Text (24):
Pretreatment with a lignan-enriched extract of Schisandra chinensis (1.6 g/kg p.o.) enhanced the hepatic glutathione status and protected against physical exercise-induced muscle damage in rats (Ko et al., Phytotherapy Research 10, 450-452, (1996)). Treatment of rats with extract from Schisandra chinensis (1.6 g/kg/day p.o.) increased the hepatic GSH level and activities of GSH reductase, G6PDH, and decreased the susceptibility of hepatic tissue homogenates to peroxide-induced GSH depletion; pretreating rats with these extracts (0.2-3.2 9/g/kg p.o.) dose-dependently protected against CCl4-induced GSH-depletion and oxidative hepatocellular damage. These data were confirmed by other experiments, showing that Schisandra chienesis extract (1.6 g/kg/day p.o.) increases hepatic GSH levels, GSH reductase and GSH-transferase activities and prevents liver damage in rats with aflatoxin- and cadmium-induced liver damage. Pretreatment of schisanhenol and schizandrin B (200 mg/kg) to mice with ethanol-induced liver peroxidation increased dismutase and catalase activity, while GSH-peroxidase activity was unaffected. Treatment of schisandrin B (3 mmol/kg/day p.o.) increased the mitochondrial GSH level and reciprocally decreased GSSG level, elevating the GSH/GSSG ratio, and increased mitochondrial GSH reductase activity. These effects were more pronounced in CCl4-intoxicated mice, providing protection against liver damage. BCNU, a specific inhibitor of GSH reductase, however, did not affect the protective activity, although it inhibited GSH reductase activity, suggesting that the enhancement of GSH reductase activity by Schisandrin B is not a primary factor of hepatoprotection.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L1: Entry 10 of 36 File: USPT Apr 24, 2001

DOCUMENT-IDENTIFIER: US 6221358 B1
TITLE: Method for lowering blood alcohol concentration by administering an extract of Rhus verniciflua

Brief Summary Text (9):
Carbon tetrachloride (CCl.sub.14) induces liver damage and is used in various animal and cultured cell tests to evaluate agents for treating hepatic disorders. CCl.sub.4 converts to a radical species, CCl.sub.3, by the action of such a metabolic enzyme as cytochrome P450 and the radical induces oxidation of the fat in the liver or the fatty acids present in the phospholipid membrane. This oxidative process is participated by oxygen to form lipid peroxides. This peroxidation process, in turn, brings about fat accumulation, lowering of protein secretion, degradation of glycogen, destruction of enzymes, and eventually, the death of liver cells. Accordingly, CCl.sub.4 -induced liver damage is used as a model in tests to evaluated liver disorders caused by ethanol (Recknagel, R. O., Pharmacol. Rev., 19, 145-208 (1967); Alpers et al., Mol. Pharmacol., 4, 566-573 (1968); Slater, Free Radicals, Lipid Peroxidation and Cancer, Academic Press, London, p 243 (1982); Chang, I. M. et al., Drug and Chemical Toxicology, 6(5), 443-453 (1989)).

Previous Doc Next Doc Go to Doc#